

Pure Memory Loss With Hippocampal Lesions

A Pneumoencephalographic Study

Osamu Muramoto, MD; Yutaka Kuru, MD;
Morihiro Sugishita, PhD; Yasuo Toyokura, MD

• A case of pure memory loss occurred suddenly following inhalation anesthesia. Examination seven years after the onset of amnesia disclosed a profound and selective impairment of recent memory without concomitant deterioration of other higher functions or evidence of other neurologic abnormalities. Pneumoencephalotomography focused on the temporal horns showed severe atrophic changes confined to both hippocampi. This finding provides a radiologic counterpart of previously reported surgical and pathological observations of bilateral hippocampal lesions in cases with relatively pure loss of memory.

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In 1957, Scoville and Milner¹ reported the observation of memory disorder with difficulties of registration following a bilateral resection of the hippocampal regions that was carried out on an intractable epileptic patient for therapeutic purposes. A particularly striking observation in this case was that despite severe memory impairment, perceptual and intellectual capacities remained intact, as manifested by normal or above-average performance of experimental tasks.

Apart from the clinical sequelae of surgical intervention, spontaneously occurring cases of memory loss with lesions involving the hippocampus on both sides are extremely rare. DeJong

et al² described a case of memory loss that occurred suddenly following cystoscopy under barbiturate and nitrous oxide anesthesia. This patient had no other impairment of intellectual function nor neurologic abnormality. Therefore, the term "pure" memory loss was applied to this state. At autopsy, there were discrete bilateral necrotic infarcts of the hippocampus. Similar cases of selective impairment of memory with detailed pathological findings were reported by Victor et al³ and Benson et al.⁴

While the relationship of focal damage in the hippocampal regions to memory disorders is now widely accepted because of these and other clinical and pathological reports,⁵ no attempt has been made as yet to correlate memory loss with radiologic alterations of the hippocampus. The case of pure memory loss herein reported is unique in that bilateral atrophic changes of the hippocampus were clinically confirmed by pneumoencephalography.

REPORT OF A CASE

A 59-year-old right-handed carpenter was well until November 1970, when, at the age of 52 years, he was admitted to a local hospital because of severe upper abdominal pain. Diagnosed as having duodenal ulcer, he was operated on while under endotracheal general anesthesia using morphine, succinylcholine chloride, and nitrous oxide. The operation was reported as uneventful, but a detailed record was not available.

During the postoperative period, members of his family noticed that he had "somewhat changed." He became forgetful about ordinary events. He would repeat the same statement to the same person on many occasions, not realizing that he had told it before. When he was discharged one month later, he was no longer able to recognize his own house. He made notes to reinforce his memory, only to find them of no avail because he could not remember

what they were meant for. In the ensuing months, his memory disorder appeared to have improved to some extent, and then remained essentially unchanged.

Although his capacity to register new information was severely impaired, he made no errors in judgment regarding his work of making furniture. He remained as skillful as before and could even teach his proficient techniques to his colleagues. His memory for remote events up to 1970 also appeared to be intact. On April 5, 1977, he was admitted to the Tokyo University Hospital for evaluation of the memory loss.

The history was not remarkable. There was no history of alcoholism. He had shown no significant personality change. The family history was not contributory.

Neuropsychological Findings

Examination disclosed an elderly man who was alert and cooperative. He was oriented to person and place but did not know the date. He underestimated his own age and showed grossly defective memory of his recent experiences. He could not recall what had happened in the preceding few hours or few minutes. In contrast, however, his memory for events that had occurred prior to the onset of amnesia appeared to be intact. He could give much detailed and accurate information about his early life. His attention was excellent and he had a digit span of seven. However, on the Supra Span Digit Storage,⁶ his maximum storage was 5.3, ie, he was unable to exceed his forward digit span. As to verbal learning and memory, the Selective Reminding Technique⁷ demonstrated that his retrieval as well as storage of verbal information were markedly impaired. In contrast to these disabilities, there was no evidence of general intellectual loss; on the Wechsler Adult Intelligence Scale he attained a full-scale IQ of 111 (verbal, 118; performance, 102). Mood and affect were appropriate. He was aware of his memory difficulty. He frequently mentioned his forgetfulness and always showed good insight, with no tendency to confabulate.

In order to evaluate the acquisition of

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From the Department of Neurology, Institute of Brain Research, Faculty of Medicine, University of Tokyo (Drs Muramoto, Sugishita, and Toyokura), and the Department of Radiology, Juntendo University Medical School, Tokyo (Dr Kuru).

Reprint requests to National Musashi Research Institute for Mental and Nervous Diseases, 2620 Ogawahigashi, Kodaira, Tokyo, Japan 187 (Dr Muramoto).

motor skills, we trained him on a mirror-drawing task five trials a day over a one-week period, in which he was required to trace a line within the narrow outlines of star-shaped pattern as reflected in a mirror. He showed steady improvement,

indicated by reduction in both time and error scores. Moreover, after a two-month interval during which he had no chance to practice, he showed almost complete retention of the level he had attained at the end of the training.

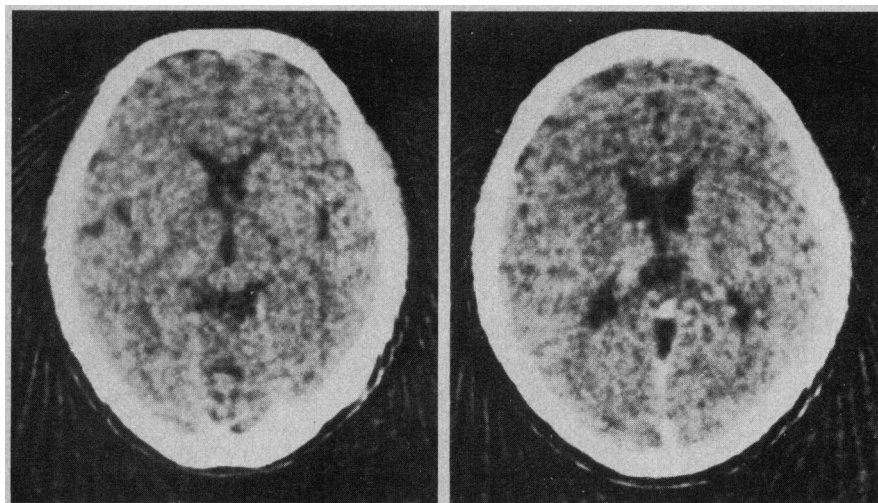


Fig 1.—Computed tomographic scan showing slight ventricular dilation.

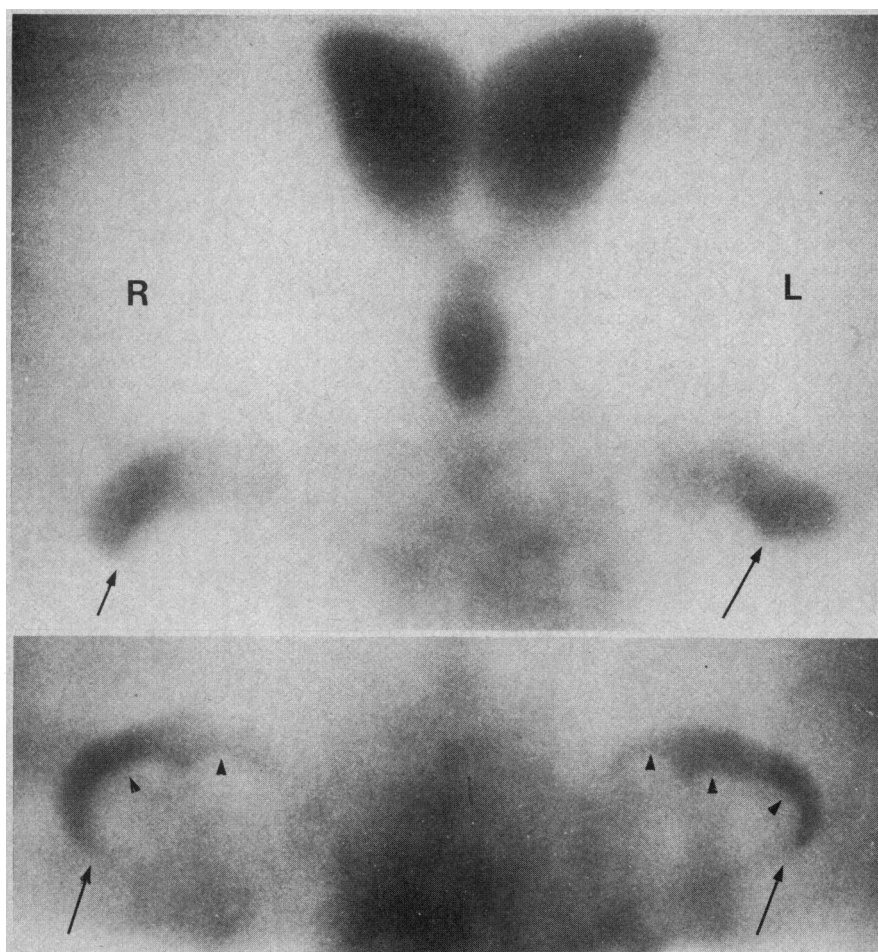


Fig 2.—Frontal cut through external auditory meatus. Top, present case. Bottom, normal control for comparison. Arrowheads indicate digitations of hippocampus. Arrows indicate lateral cleft. Note severe shrinkage of hippocampi and marked widening of lateral cleft.

Examination Findings

Physical and neurologic examinations gave entirely normal findings except for the following. There was some limitation of range of motion at the neck and moderate weakness and atrophy, especially on the right, of proximal portion of the upper limbs. Fundoscopic examination disclosed moderate arteriosclerotic changes of the retinal vessels. He was slightly hypertensive (blood pressure, 150/96 mm Hg).

Plasma lipid levels in the fasting state were as follows: total cholesterol, 272 mg/dl; triglyceride, 170 mg/dl; and free fatty acid, 1,400 mEq/liter. Plasma lipoprotein pattern on agarose-gel electrophoresis showed moderate increases in β -lipoprotein and pre- β -lipoprotein, which is characteristic of hyperlipoproteinemia type IIb according to the World Health Organization classification of hyperlipoproteinemia.⁸ Blood B₁ and B₁₂ levels were within normal limits. He was not diabetic. The remaining laboratory values were entirely within normal limits. Skull roentgenogram, electrocardiogram, and EEG showed no abnormalities. Computed tomographic (CT) scan (Fig 1) was interpreted as normal except for slight ventricular dilation. Roentgenograms of the cervical and lumbar spine including laminagraphy showed marked spondylotic changes that were compatible with the clinical finding. Results of CSF study were unremarkable

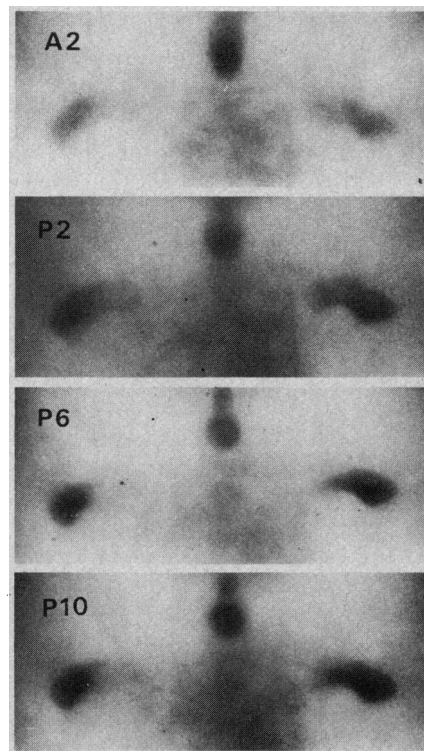


Fig 3.—Frontal cut, serial tomograms. Tomographic cutting was made with 2-mm thickness; midpoint of both external auditory meatus was chosen for reference point. A2 indicates 2 mm anterior reference point; P2, 2 mm posterior to reference point.

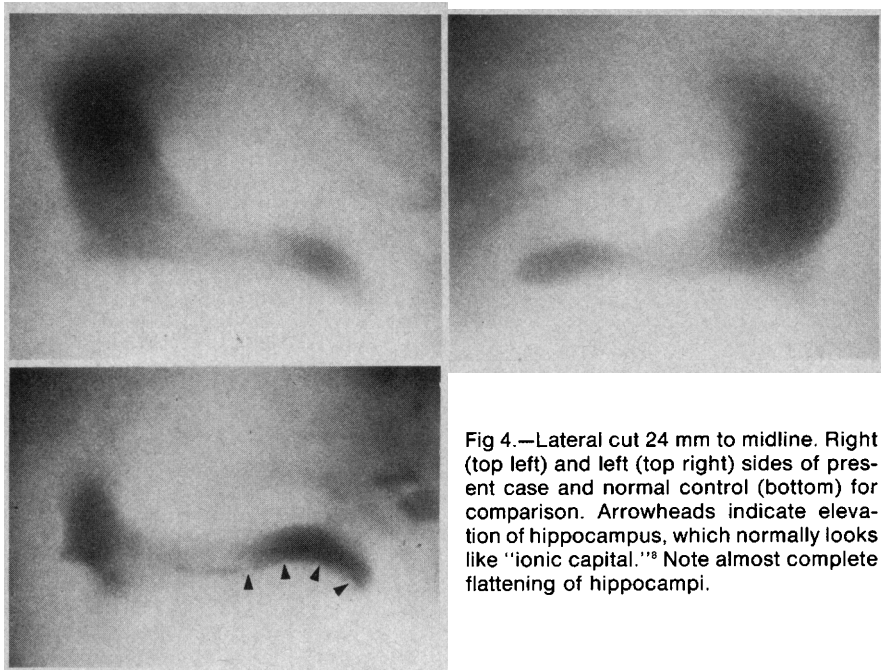


Fig 4.—Lateral cut 24 mm to midline. Right (top left) and left (top right) sides of present case and normal control (bottom) for comparison. Arrowheads indicate elevation of hippocampus, which normally looks like "ionic capital." Note almost complete flattening of hippocampi.

except for a protein concentration of 90 mg/dl. Angiographic investigations of all four cerebral vessels were interpreted as normal except for moderate arteriosclerotic changes involving multiple intracranial arteries.

Pneumoencephalographic Findings

In an effort to obtain a specific clue to the pathogenesis of this patient's amnesia, a pneumoencephalographic study focused on the temporal horns was performed by the lumbar route.

On the frontal tomograms (Fig 2), the fourth as well as the third ventricles appeared slightly dilated (Evans index, 0.26). Both temporal horns, especially on the left side, were considerably enlarged. The lateral cleft, or diverticle of subiculum, which normally exhibits an acute angle, was widened and blunted. This widening is thought to be the result of the decrease in volume of the Sommer's sector.⁹ The hippocampus, which normally has a prominent bulge on the floor of the temporal horn, was so severely shrunken that it was reduced to approximately one third of normal size. A normal pattern of digitations on the surface of the hippocampus¹⁰ was hardly shown in any slice. These changes of the hippocampus were roughly symmetrical but more prominent on the

left side. Serial tomograms (Fig 3) demonstrated the extent of the severe atrophic changes of the hippocampi. On the lateral tomograms (Fig 4), the elevation of the hippocampus at the floor of the temporal horn, which normally has a characteristic appearance of "ionic capital",¹⁰ was slightly traceable on the right side, while on the left, this was hardly seen in any slice and the hippocampus appeared almost entirely flattened out.

In summary, pneumoencephalographic studies showed a high degree of bilateral atrophy of the hippocampus with slight dilation of the third and fourth ventricles.

COMMENT

The profound memory loss in the present patient developed shortly after inhalation anesthesia, and the subsequent course of his disability was not progressive. Examination on admission disclosed generalized arteriosclerotic changes of cerebral vessels as well as hyperlipoproteinemia type IIb. These underlying conditions are quite similar to those of the case described by DeJong et al.² Taking these into account, one can assume that permanent cerebral damage oc-

curred during a period of relative hypotension or hypoxia that accompanied the anesthesia.

The pneumoencephalograms demonstrated that, while there existed slight and generalized dilation of the ventricles, the major and disproportionately severe changes were localized in both hippocampi. These changes therefore seem to correspond to the previously reported autopsy findings of discrete lesions of the hippocampi in cases of memory loss. To our knowledge, pneumoencephalographic abnormality of this kind in amnesic patients has not been described previously.

It is noteworthy that, despite the serious inability to learn and to retain new facts, the patient's memory for his premonitory acquired skills for woodworking has been unaffected. Furthermore, he could indeed acquire new motor skills, as manifested by the performance of a mirror-drawing task. This fact is in accordance with Milner's¹¹ report that his patient's memory loss left technical skills intact. Corkin¹² confirmed this by experimental procedures, suggesting the possibility that motor learning can be mediated by brain structures other than the hippocampus.

In the present case, computerized tomographic (CT) scan failed to provide information about the lesions mentioned above. We therefore believe that, despite the growing importance of the CT scan, pneumoencephalography is still helpful to detect small lesions located close to the ventricles and will play a continuing role of importance in the evaluation of some neurologic diseases.⁹ Among others, an emphasis should be placed on the role of pneumoencephalography in detecting hippocampal lesions in the evaluation of amnesic patients.

Samuel M. Chou, MD, PhD, Department of Neuropathology, West Virginia University Medical Center, Morgantown, and Hideo Sugita, MD, Department of Neurology, Institute of Brain Research, University of Tokyo, reviewed the manuscript.

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